Remarks

The change made at page 40, line 4, was made to correct the volume number of the reference described. The author's name, the journal title, the issue and page numbers were all correctly described, and from that information the correct reference could be easily found.

Conclusion

The Examiner is invited to contact Applicants' Representatives at the below-listed telephone number, if there are any questions regarding this Preliminary Amendment or if prosecution of this application may be assisted thereby.

The Examiner is invited to contact Applicants' Representatives at the below-listed telephone number, if they can be of any assistance during prosecution of the present application.

CERTIFICATE UNDER 37 C.F.R. 1.8:

The undersigned hereby certifies that this paper is being deposited in the United States Postal Service, as first class mail, in an envelope addressed to: Assistant Commissioner for Patents, P.O. Box 2327, Arlington, VA 22202, on this 23 day of May, 2002.

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May 23, 2002
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Respectfully submitted for
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APPENDIX A - SPECIFICATION/CLAIM AMENDMENTS INCLUDING NOTATIONS TO INDICATE CHANGES MADE

Applicants : James Travis, et al.
Serial No. 10/030,330
Filed: October 19, 2001 (Int'l. Filing Date: April 20, 2000)
Attorney Docket No.: 235.0021 0101
Title: A POLYPEPTIDE HAVING AMIDOLYTIC ACTIVITY FOR A SERPIN

Amendments to the following are indicated by underlining what has been added and bracketing what has been deleted. Additionally, all amendments have been shaded.

In the Specification

The paragraph beginning at page 39, line 21, has been amended as follows:

The regulating inhibitor of HNE is α_1-PI, a plasma protein which forms a complex with this proteinase and is rapidly removed from the circulation and degraded (Mast et al., J. Biol. Chem., 266(24):15810-15816 (1991)). The inhibitor, however, can itself be inactivated by either oxidation at its reactive site or by proteolytic cleavage by nontarget proteinases within the RSL region (Travis et al., Annu. Rev. Biochem., 52:655-709 (1983)), and it is believed that both mechanisms occur during the development of emphysema. Certainly, the high levels of active HNE in the GCF, despite the presence of α_1-PI, would suggest that parallel mechanisms for inhibitor inactivation may be also occurring in periodontal disease (Travis et al., Annu. Rev. Biochem., 52:655-709 (1983)). In this respect, it has been reported that whole cells or culture supernatants from *P. gingivalis* are capable of proteolytically inactivating α_1-PI (Grenier, Microbiology, 142:955-961 (1996); Carlsson et al., Infect. Immun., 43(2):644-648 (1984)), although it is clear that this is not due to any of the gingipain –R or –K forms since the inhibitor contains no basic residues within its RSL (Potempa et al., J. Biol. Chem., [274]273(34):21648-21657 (1998)). Thus, another proteinase(s) must be involved in this process, and it is likely that periodontain serves this purpose.